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REVIEW

An Analysis Comparing Open Surgical and Endovascular Treatment of Atherosclerotic Renal Artery Stenosis

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Abstract *Objective:* Endovascular revascularization in atherosclerotic renal artery stenosis (ARAS) has dominated during the last 15 years with surgery relegated mostly to back-up for failed endovascular procedures. This study examines the available outcome evidence to determine what role open surgery should have in comparison to endovascular treatment in the management of ARAS.
Method: Of 183 papers listed in PubMed, the USNLM and the Cochrane library, (1975–2004) 47, dealing with outcomes of surgical and endovascular treatments (evidence levels 2b and 3) were selected. Endovascular included 1750 patients in 16 prospective non-randomised (PNRT) and 5 retrospective (RET) studies. Surgical included 2314 patients in 4 PNRTs and 17 RETs. Outcome data were subjected to meta-regression analysis weighted according to the inverse variance method.
Results: Mean maximum ages were 79.4 yrs (SD 6.9) for surgical and 83.6 yrs (SD 3.8) for endovascular studies. Primary technical success was similar. Endovascular patency declined by 0.26%/month (95% CI: 0.04–0.48, $p = 0.03$). Surgical studies showed greater improvement for hypertension control by 21% (95% CI: 9–33%, $p = 0.001$) and for renal function by 34% (95% CI: 18–54%, $p < 0.001$), as well as a higher creatinine reduction by 32 μmol/L (95% CI: 7–57 μmol/L, $p < 0.014$). A higher excess surgical mortality, 3.1% (95% CI: 1.8–4.4%, $p < 0.001$) became insignificant, 0.18% (95% CI: 0.7–1.1, $p = 0.70$) when concomitant aortic surgery was excluded.
Conclusion: This data shows a marked and durable clinical benefit for surgery. These findings question the endovascular predominance in intervention in ARAS and highlight the need for a carefully designed prospective randomised comparison to define the roles of endovascular and surgical renal revascularization.

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Introduction

Revascularization in the management of atherosclerotic renal artery stenosis has undergone a series of changes over the past 4 decades.

After Goldblatt's experiments in 1934[1]and Leadbetter's report of the first cure of hypertension by surgical renal artery revascularization in 1938[2] a direct causal relationship was inferred. Since then the evolution of our knowledge of the pathophysiology of renal artery stenosis and modern medical treatment together with the asynchronous developments in diagnosis, in surgery, and endovascular techniques has taken management of the stenotic renal artery through a surgical phase then an endovascular phase. After recent input from key meta-analyses,[3,4] the role of endovascular revascularization is being assessed in favour of or in tandem with medical management in trials such as STAR, NITER, CORAL, RAVE and ASTRAL. Indeed the trend seems to be to avoid intervention altogether however though these studies include endovascular intervention in the treatment process, they mostly fall short of investigating the value and effect of restoring and maintaining patency on their outcomes. Management of ARAS has traditionally fallen within the realm of nephrologists with largely technical input from surgeons and now mostly endovascular interventionalists. Cardiologists have recently questioned the role of ARAS in the management of CCF[5,6] especially in the absence of left ventricular dysfunction and coronary ischaemia. The US Renal Data Systems (2005) reported a 2.1% incidence of ESRD (>9000 patients) secondary to renal artery stenosis/occlusion with a 36.7% first year mortality. The contribution of ARAS to CCF has yet to be recognized and measured. Unfortunately, ARAS is not an isolated causative lesion of a direct clinical manifestation as fibromuscular dysplasia may be of hypertension. It is one contributing factor in the interplay of the hypertension, renal failure and congestive cardiac failure triad within the background of the wider systemic manifestations of atherosclerosis in our increasingly diabetic, hyperlipidaemic and aging population.

Despite advances in diagnostics, therapeutics and management, the literature on ARAS reflects a failure to reach clear consensus on the indications for revascularization. Changes in the decision process in ARAS management have been largely driven by innovations, and trends in reporting and treatment costs, with improvement of hypertension control and renal function, or renal dialysis salvage being the commonest targets. Much of the published literature falls short of a holistic long-term approach in terms of outcome and patency, improved quality of life and survival benefit.

The current aversion to surgical revascularization is understandable in this largely elderly and often unfit cohort of patents but focused clinical evidence to support this shift does not exist. Current management is based more on clinical bias rather than evidence and the authors are concerned that as a consequence, a minority of patients that would benefit from surgical revascularization are not being offered this treatment option. The major arguments against surgical revascularization include the following:

1. The level of co-morbidity in these patients forbids the use of anaesthetic dependent surgery.
2. Outcome studies for surgical revascularizsation only include younger fitter patients.
3. Endovascular and surgical procedures confer similarly successful revascularization.
4. Endovascular and surgical procedures are similarly successful in terms of outcome benefit.
5. The mortality linked to surgery is too high to justify it as a treatment option.

In this review we chose to analyse the clinical evidence that is available in the literature and have addressed the issues of age, co-morbidity, technical success and patency, survival, outcome benefit in hypertension and renal function and procedure-related mortality. We found a wide heterogeneity in published papers in terms of indications for intervention and in the types of outcomes and data presented to depict success or otherwise of outcomes. This has unfortunately limited the study to observational retrospective and prospective non-randomised studies with a complete absence of Level 1 data.

Methods

Search Strategy: PubMed, the USNLM and the Cochrane Library were searched for studies dealing with clinical outcomes of surgical and endovascular revascularization for ARAS. Search terms were sought in title, abstract and keyword fields and included the following: atherosclerotic renal artery stenosis, renal artery revascularization, renal artery stenting and angioplasty, surgical revascularization, renovascular hypertension, ischaemic nephropathy and renal failure. 183 papers were initially retrieved in abstract form and scanned. Abstract selection: abstracts dealing with outcomes of endovascular and surgical revascularization targeting improvement in hypertension control and renal function were selected. Exclusion criteria included other renal aetiologies, lack of an English language version and other outcome targets. Remaining papers were then retrieved and reference lists were examined for other relevant publications.

Further exclusion criteria were applied: following Nordmann's recommendations in the 2003 Cochrane review[4] endovascular revascularization studies were limited to stented studies. Studies selected were those in which the data under examination could be extracted and pooled (Table 1) and mainly fell into the category of prospective and retrospective cohort studies. Fields examined included: demographic data, co-morbidity, length of follow-up, technical success, benefit to hypertension and renal function, change in mean arterial blood pressure (MAP), change in serum creatinine levels, mortality, patency and 5-year survival. MAP was calculated from basic systolic and diastolic pressure data in the papers. Serum creatinine levels were converted to SI units where other units were used. Creatinine clearance would have been a better measure of renal function however this was reported in very few of the papers and only used as an outcome measure in 2 endovascular and 1 surgical papers.

Other consideration was given to analyzing the impact of whether revascularization was unilateral or bilateral, the relationship to pre-procedural degree of stenosis, the

Table 1 Papers included in this study: number of patients in each paper, mean age in years and % males. Where mean age was not available, age range was included.

Surgical papers					Endovascular papers				
Author	Year	Patients	Mean age	% males	Author	Year	Patients	Mean age	% males
Pechan BW[9]	1979	56	n/a	58.9	Rees CR[10]	1991	28	66	46.4
Novick AC[11]	1983	51	61	74.5	Raynaud A[12]	1994	18	58	55.6
Rieder CF[13]	1984	28	62	85.7	Dorros G[14]	1995	76	67	52.6
Chibaro EA[15]	1984	36	61	55.6	van de Ven PJ[16]	1995	24	66	54.2
Sicard GA[17]	1985	20	59	65.0	Iannone L[18]	1996	63	70	49.2
Novick AC[19]	1985	13	(56–74)	61.5	Rundback JH[20]	1996	20	70	55.0
Torsello G[21]	1990	326	58	67.8	Boisclair C[22]	1997	33	63	57.6
Stansby G[23]	1992	12	65	33.3	Harden PN[24]	1997	32	67	n/a
Bredenberg C[25]	1992	66	63	n/a	White CJ[26]	1997	100	67	42.0
Libertino JA[27]	1992	97	62	46.4	Dorros G[28]	1998	145	67	50.3
Gill IS[29]	1993	13	59	76.9	Gross CM[30]	1998	30	66	63.3
Reilly J[31]	1994	48	66	31.3	Rundback JH[32]	1998	45	70	51.1
Chaikof E[33]	1994	50	66	74.0	Tuttle KR[34]	1998	129	71	48.8
Van Damme H[35]	1995	23	63	69.6	Henry M[36]	1999	210	68	66.2
Clair DG[37]	1995	43	68	58.1	Rocha-Singh KJ[38]	1999	140	67	41.9
Fergany A[39]	1995	171	62	54.9	Rodriguez-Lopez JA[40]	1999	108	72	59.2
Hallett J[41]	1995	304	68	71.7	Watson PS[42]	2000	33	72	51.5
Cambria RP[43]	1996	139	67	53.2	Lederman RJ[44]	2001	300	70	52.0
Steinbach F[45]	1997	220	60	57.7	Bush R[46]	2001	73	68	45.2
Cherr G[47]	2002	500	65	49.2	Henry M[48]	2003	56	66	57.1
Marone LK[49]	2004	96	70	48.8	Zhang Q[50]	2003	87	71	60.9

association with aortic disease and procedures, and the salvage from and progression to dialysis. Though all surgical and many endovascular papers gave a detailed breakdown of the lesions being treated, very few correlated this to outcomes.

Data were analysed using SPSS® version 15 and Stata version 10. Descriptive statistics refer to means of aggregated outcomes. To assess the effect of aortic repair on mortality and the benefits of the treatment modalities on hypertension and renal function and patency analysis, meta-regression analysis was carried out weighted according to the inverse variance method, giving more weight to larger studies. For outcomes represented as percentages (e.g. mortality), the variance for a binomial distribution was used, based on the percentage itself and the sample size for the study. For means (MAP and creatinine), the intention was to divide the standard deviation squared divided by the sample size. However the standard deviation was reported only for a minority of studies. For those studies, the standard deviation value was similar between the studies. Therefore in the meta-regression analysis, weighting was done only according to the sample size.

In meta-regression, the intervention was the chief independent variable, but where the post-procedural mean MAP or creatinine was the dependent variable, pre-procedural means were included as a covariate.

To investigate the impact of study size on the differences in outcome between surgical and endovascular studies, an interaction term was added to the weighted regression model. Study size was dichotomized as to whether the studies had more or less than 40 patients. A similar analysis of interaction with type of study compared the effect in prospective non-randomised studies with retrospective studies.

Little evidence of interaction was found for type of intervention either with size or type of study. The only evidence of interaction was for improvement of hypertension according to size of study: in small studies there was no difference between surgical studies and endovascular studies, in larger studies, surgery was found to be superior (p for interaction $= 0.005$). Despite the detailed nature of some of these analyses we acknowledge that the heterogeneity in the data extracted from these papers weaken the evidence base and prevent us from drawing robust conclusions.

Results

The selection processes yielded a total of 21 endovascular and 21 surgical studies including a total of 4064 patients: 1750 endovascular patients in 16 prospective non-randomised (PNRT) and 5 retrospective (RET) studies and 2314 surgical patients in 4 PNRTs and 17 RETs. Significant stenosis was defined as being >50%, >60%, >70% and >75% in 5, 4, 9 and 1 endovascular papers and >50%, >60%, >70%, >75%, and >80% in 2, 3, 1, 9 and 1 surgical papers respectively. 2 further surgical papers included only 'high grade' stenoses.

Age and co-morbidity

The first issue addressed was whether endovascular studies included sicker and older patients. There was broad similarity in age ranges across all the studies. Mean age was 67.8 years for endovascular, and 62.5 for surgical studies. Mean maximum age was 83.6 years for endovascular and 79.4 years for surgical studies. Medians of the age ranges

Table 2 Co-morbidity data is similar across endovascular and surgical studies.

Co-morbidity	Surgical papers	Prevalence (%)		Endovascular papers	Prevalence (%)	
		Mean	Median		Mean	Median
Ischaemic heart disease	12	53.4	52.9	15	59.9	60
Cerebrovascular disease	10	32.5	30	9	26.8	29
Diabetes	10	13.6	15	17	25.5	25.5
Peripheral vascular disease	13	58.7	56.4	13	48.6	48
Smoking	6	66	68.5	14	49.1	49.5

would have given a clearer picture of the age distribution however this data was not available most papers.

No data was available about the state of health of patients at the time of intervention and the reporting of co-morbidities varied among different papers. Nonetheless distribution and prevalence of co-morbidities were similar for both modalities (Table 2).

Follow-up

Difference in length of follow-up was notable. Mean of average follow-up was 41.7 months (SD 27.5) in surgical and 15.3 months (SD 9.6) in endovascular studies. Time-points of measurement of outcome data indicating effects on hypertension and renal function however were not equivalent to length of follow-up in most of the papers. The mean time point for endovascular papers was 7.1 months, median 6 months.

The mean time point for surgical papers was 25.1 months, median 22 months. This difference changes the impact of the surgical outcome data such that surgical outcomes pooled in this paper could be seen as sustained long-term results. Combined time point and outcome data including size of study population are shown in Fig. 1.

Technical success

Technical success was minimally but non-significantly lower in surgical studies by 0.09% (95% CI: 0.62−0.80, $p = 0.8$). The surgical data however included the results of combined renal and aortic procedures with those of isolated renal revascularization.

Patency

This study was unable to compare maintenance of patency beyond the initial technical success since there was insufficient patency data in surgical studies for analysis. 2 individual papers quoted 98% and 92% patency at 36 and 60 months respectively. Patency data from 13 endovascular papers were for the first year of follow-up with two further papers quoting patency up to 36 months and one paper up to 60 months. Analysis of this data showed that stent patency declined by 0.26% per month (95% CI: 0.04−0.48, $p = 0.03$), equivalent to a 3% absolute decrease in patency per year.

Survival

4 out of 26 endovascular papers supplied survival data (Table 3), which was insufficient for analysis. 10 surgical papers supplied 5-year survival data ranging from 48 to 94% (Fig. 2) and using denominators for each study, gave a combined estimate of 68%.

Hypertension

There was a 21% higher rate of improvement in hypertension in surgical procedures compared to endovascular (95% CI: 9−33%, $p = 0.001$). The definitions of improvement included normalization of blood pressure to diastolic pressure $\leq$90 mmHg, a reduction in medications to maintain diastolic pressure $\leq$90 mmHg, a reduction in diastolic pressure by 15−20 mmHg or reduction in diastolic pressure by 15%. Data for percentage improvement and deterioration are depicted in Fig. 1. Measurements were generally taken at later time-points after surgical than after endovascular procedures suggesting that improvement resulting from surgical revascularization is sustained for longer periods.

Differences between pre-procedural and immediate post-procedural blood pressure have been simply presented in Table 3 and Fig. 3a. The pre-procedural mean blood pressures are similar in both the surgical and the endovascular papers however there is a greater reduction in post-procedural blood pressure in surgical papers.

These findings were further analysed to correct for difference in study size and for variation in pre-procedural blood pressure between the papers. This revealed a non-significant 0.8% lower rate of deterioration in hypertension in surgical procedures (95% CI: −0.7 to 2.2, $p = 0.28$). A weakly significant greater decrease in systolic pressure by 5 mmHg (95% CI: −3−12 mmHg, $p = 0.087$) and diastolic by 4 mmHg (95% CI: 1−8 mmHg, $p = 0.012$) was found, after adjustment for pre-procedural blood pressures. The difference in post-procedural MAP was 3.1 mmHg (95% CI: −1.0−7.3, $p = 0.13$), being lower in the surgical group after adjustment for pre-procedural MAP (Fig. 3a).

It is important to distinguish between describing improvement in hypertension in terms of blood pressure measurements and in terms of the applied definitions of improvement in hypertension control which include reduction in number of medications in addition to actual pressure changes. Also, post-surgical blood pressure fluctuates widely in response to inadequate pain control and other noxious stimuli.

Renal function

There was a 34% higher rate of improvement in renal function in surgical procedures compared to endovascular

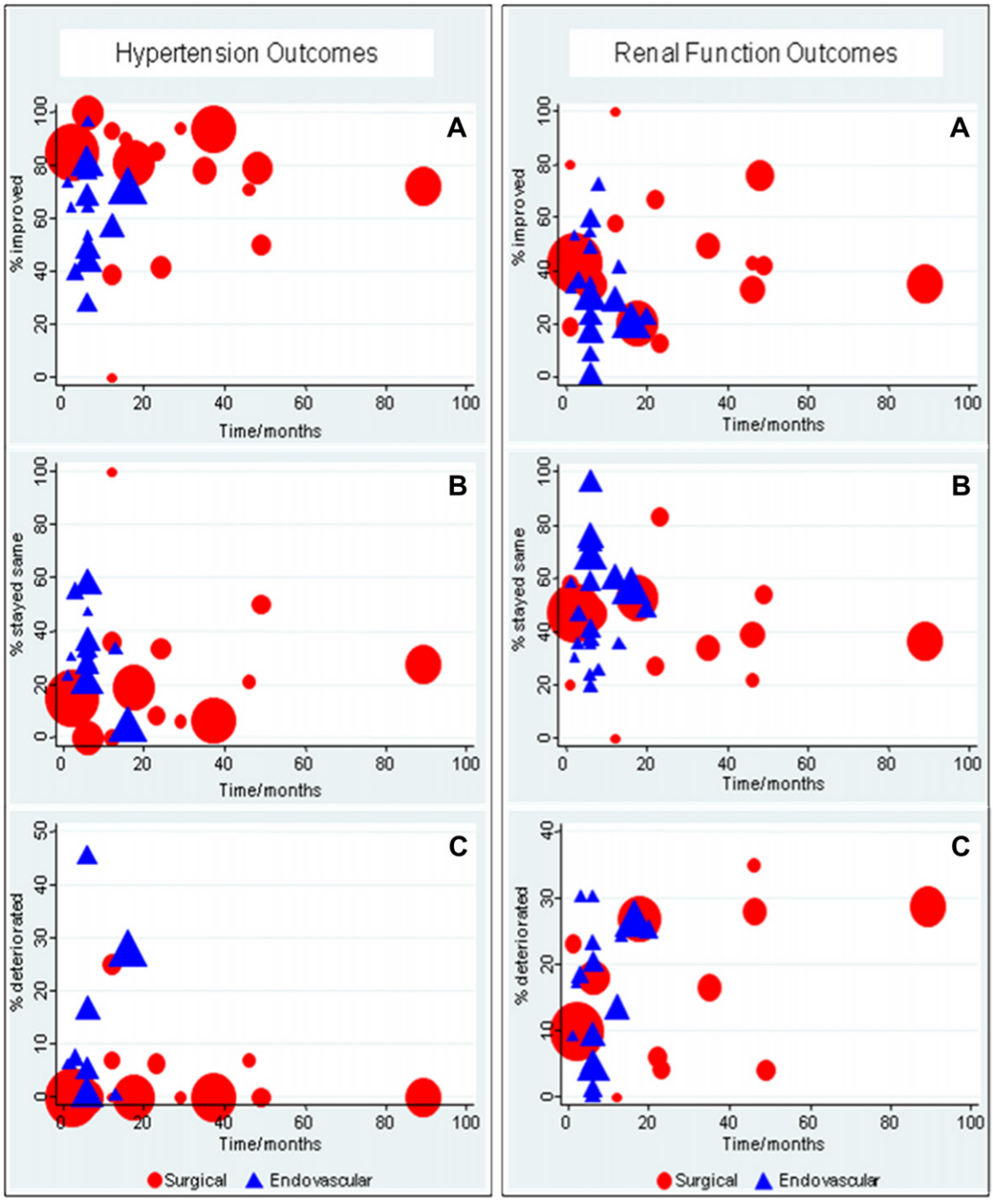

Figure 1 Scatter-graphs of hypertension or renal function improvement (A), no change (B) and deterioration rates (C) against average time-points of measurement for surgical (red circles) and endovascular (blue triangles) studies. The sizes of points are proportional to study size. Longer surgical time-points suggest sustained longer-term outcome benefit.

(95% CI: 1.8—54%, $p < 0.001$) and a significant 8.8% lower rate of deterioration in renal function in surgical procedures (95% CI: 2.6—14.9, $p = 0.006$). Definitions of improvement in renal function varied. One surgical paper required a 20% increase in eGFR (estimated Glomerular Filtration rate) and 2 endovascular papers require a 15% and 20% improvement in creatinine clearance respectively. Remaining papers used serum creatinine levels as an outcome measurement. Improvement was not defined in 13 papers. 11, 5 and 2 papers required a decrease in creatinine by 20%, 15% and 10% respectively. 3, 1 and 2 papers required a decrease in creatinine of at least 17.5 μmol/L, 35 μmol/L and 80 μmol/L respectively.

To give another perspective, the difference between pre-procedural and immediate post-procedural creatinine levels is presented in Table 3 and Fig. 3b. It is immediately evident that surgical papers studied populations with worse renal function than endovascular papers and the latter seem to have actually worsened renal function overall.

Table 3 Pre-procedural blood pressure and serum creatinine measurements and poet-procedural reductions. The negative value in creatinine reduction denotes a relative deterioration however average pre-op creatinine was higher in surgical papers.

Parameters assessed	Surgical papers			Endovascular papers		
	Mean	Median	SD	Mean	Median	SD
Pre-op systolic BP (mmHg)	178.6	176.0	13	172.9	169	11.1
Reduction in systolic BP (mmHg)	33.0	34.0	13.8	23.0	19.3	15.9
Pre-op Diastolic BP (mmHg)	97.2	96.0	5.8	93.2	87.5	10.8
Reduction in diastolic BP (mmHg)	16.1	16.0	7.9	10.8	9.0	6.3
Pre-op MAP (mmHg)	123.0	121.7	9.9	119.3	116.5	9.0
Reduction in MAP (mmHg)	19.8	19.0	12.2	12.4	9.5	7.5
Pre-op creatinine (μmol/L)	259.4	239.0	71.4	158.1	150	29.6
Reduction in creatinine (μmol/L)	85.3	62.0	60	−2.2	−2.0	11.9

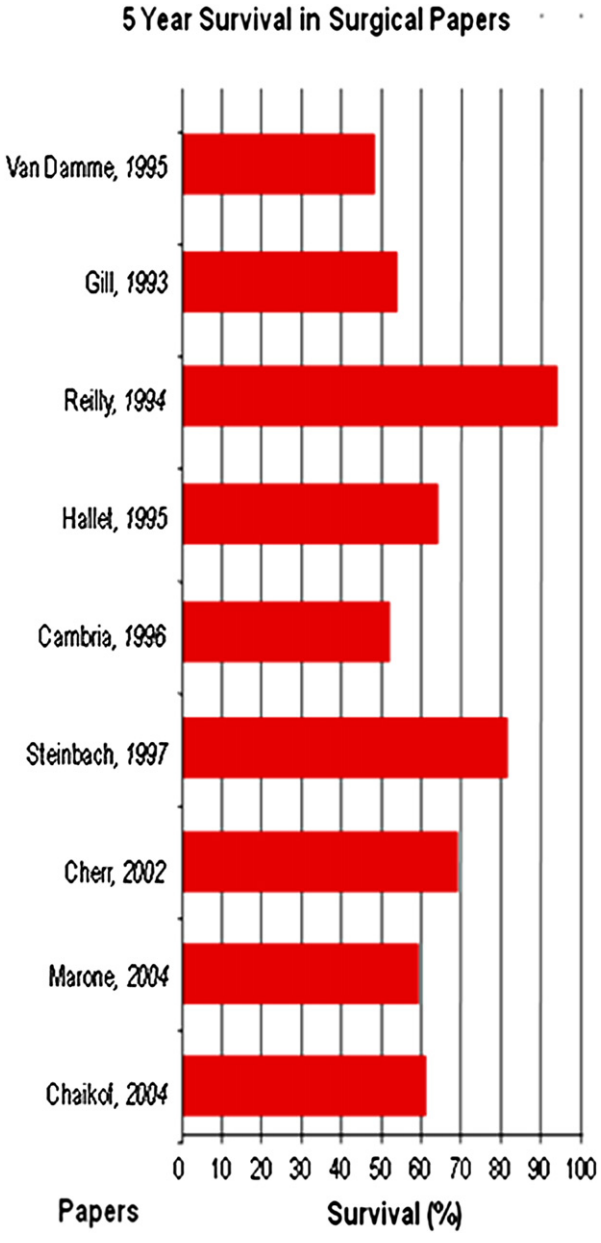

Figure 2 5-year mortality for surgical studies.

As with the blood pressure findings, this data was further analysed to correct for difference in study size and for variation in pre-procedural blood pressure between the papers. A significantly greater creatinine reduction of 54 μmol/L (95% CI: 28−80 μmol/L, $p < 0.001$) was found after surgical treatment. The difference in post-procedural creatinine was 32 μmol/L (95% CI: 7−57, $p = 0.014$), being lower in the surgical group, after adjustment for pre-procedural creatinine.

Mortality

Initial comparison of mortality data showed a significant 3.1% excess procedure-related (30 day) mortality for surgical treatment (95% CI: 1.8−4.4, $p < 0.001$). Excess mortality denotes the average difference in mortality between surgical and endovascular studies. Subsequent inspection of the papers showed that several surgical papers incorporated the results of more ambitious surgery such as concurrent abdominal aortic aneurysm repair and surgery for aortic occlusive disease which increase operative mortality. These procedures constituted 27.7% of total surgical procedures. Isolated renal revascularization included a variety of procedures: aorto-renal bypass with vein or prosthetic grafts (23%), extra-anatomic bypass (61%), endarterectomy patch angioplasty (14.6%) and bench reconstruction and re-implantation (1.4%).

There was sufficient data to separate results for combined aortic and renal procedures from isolated renal artery revascularizations. Combined procedures conferred a significant excess surgical mortality of 6.5% (95% CI: 3.8−9.3, $p < 0.001$) compared to isolated renal revascularization Comparison of endovascular with isolated renal artery surgical revascularization revealed an insignificant 0.18% excess mortality compared to endovascular mortality (95% CI: 0.7−1.1, $p = 0.7$) (Table 4).

Discussion

This analysis of the available data suggests that surgical revascularization is a valuable treatment which has better outcomes and durability than stent angioplasty. The current limitation of surgery as a largely salvage measures in technical failure of endovascular procedures can be challenged by these results.

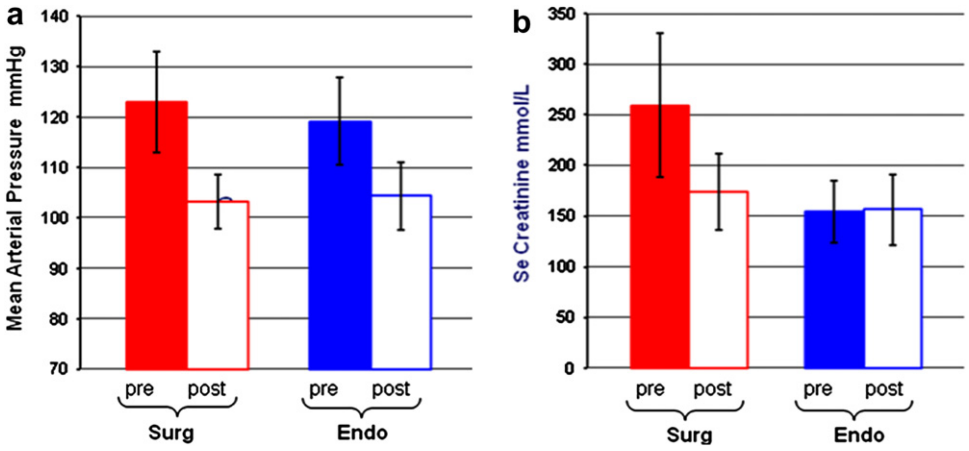

Figure 3 Graphs showing the pre- and post-procedural MAP (a) and serum creatinine (b) demonstrating advantage for surgical studies and overall deterioration in post-procedural renal function in endovascular studies.

From the outset we have recognized the major problem with this analysis namely, the lack of high quality clinical data to allow comparison of the two modalities. All included studies are observational with variation in the selection criteria of patients in different studies in terms of severity of stenosis, renal length, renal function and severity of hypertension. Additionally several publications were excluded because the data were not accessible in compatible format to allow analysis. We have presented a pooled estimate of difference between surgical and endovascular procedures for each outcome, to provide a useful summary of this data from a large number of studies from which to infer the relevant clinical importance of surgical and endovascular renal revascularization.

During the time period covered by this study, there has been progress in stent technology, pharmacology and non-interventional management of ARAS. However there has also been significant development in pre-operative cardiovascular optimization, selection, anaesthesia and peri-operative management, making surgery a lower-risk option in comparison to 15—30 years ago when most of these surgical series were undertaken. The two major shortcomings in studies investigating the outcomes of revascularization have consistently been a lack of long-term follow-up of patency and its correlation to changes in outcomes. It has been increasingly recognized that revascularization of a main stem renal artery does not improve outcomes when there is irreversible end-organ dysfunction in the form of atherosclerotic nephropathy and hypertensive nephrosclerosis. Prediction of renal parenchymal injury by applying broad selection criteria such as renal length, limits on serum creatinine levels and the presence or otherwise of proteinuria have not yielded uniform outcome responses. The use of renal biopsy for the diagnosis and scoring of histopathological damage in atherosclerotic nephropathy reported by Wright,[7] showed consistent correlation between scoring severity and renal functional prognosis. This practice has not been widely adopted because of the risk of damage to already compromised kidneys, additional costs and the invasive nature of this procedure. One of the outstanding findings in this paper is the difference in improved outcomes in terms of hypertension control and renal function after surgery despite the fact that these improvements were measured at later time-points than in endovascular studies. This also suggests that the benefits from surgical outcomes are sustained for longer periods. Endovascular revascularization is confirmed as giving less functional improvement than expected from the excellent technical success rates. The main theory to explain this deficiency is atheroembolism from the considerable trauma to the vessel walls both during catheterization and angioplasty and stenting. The recently introduced protection devices still allow passage of showers of microemboli up to 100 μm in size, enough to block the glomerular pores which are only 8 μm. Another possible phenomenon is the release of tissue factor, other procoagulant inflammatory cytokines and thrombin by the inflammatory response to the angioplasty procedure. Although these factors could also complicate surgical revascularization, this procedure requires the use of clamps and routine flushing of vessels to remove any thrombus or particulate matter prior to completion of an anastamosis, though clamping itself potentially induces ischaemic injury. Yet another plausible theory is the effect of the destruction of the sympathetic innervations along the renal artery during surgical dissection.

Table 4 Surgical mortality has been disadvantaged by the inclusion of aortic aneurysm repair. The separate mortality for surgical isolated renal revascularization is not significantly higher than for endovascular procedures.

	Increased mortality	95% CI	*p* value
All surgical vs endovascular procedures	3.1%	1.8—4.4	<0.001
Surgical revascularization with vs without simultaneous aortic repair	6.5%	3.9—9.3	<0.001
Isolated surgical revascularization vs endovascular revascularization	0.18%	0.7—1.1	=0.70

A second significant finding is that once mortality from concomitant aortic repair is excluded, no significant difference in mortality was found between surgical and endovascular repair. This lack of mortality benefit for endovascular revascularization may be explained by case-mix with higher risk patients selected for stent angioplasty rather than surgery. Nevertheless, this analysis shows that surgery focused purely on renal revascularization carries an acceptably low operative mortality. Surgeons have several options for revascularising the renal arteries which avoid cross-clamping of the aorta such as aorto-renal bypass and renal artery endarterectomy,[8] where the aorta is side-clamped, extra-anatomic bypass and rarely extra-corporeal reconstruction with autotransplantation. The extra-anatomical bypass is favoured by most surgeons because of the subcostal incision with its lower morbidity and the complete avoidance of the aorta.

This study shows that for suitable patients surgery could be a significantly better option in terms of improved and durable control of hypertension and improved renal function. However there is no Level 1 evidence to support this conclusion. As a result of the almost universal preference for stent angioplasty, vascular surgeons in the UK and much of Europe are no longer a regular part of the clinical team treating renovascular disease. This and the resultant decrease in surgical expertise means that a prospective randomised comparison of surgical and endovascular revascularization is unlikely to happen. The results of our analysis show that such a comparison is needed to define the roles of the two modalities in treating renovascular disease. It is likely that the two modalities are complimentary with stent angioplasty the treatment of choice in sicker patients with a short life expectancy and surgical reconstruction avoiding the aorta, the choice for younger, fitter patients. We have already implied that the decision process is complex and crucially depends on multi-disciplinary interaction to optimize outcomes. There is clearly an important role for vascular surgery and based on the results of this analysis we suggest an algorithm for the management of patients with atherosclerotic renovascular disease (Fig. 4). We recognise that this algorithm is not based on unshakable Level 1 evidence however it may serve as a guide in the clear absence of such evidence and would be the ideal basis for a randomised controlled study especially if a medical arm was included.

Finally there is a lack of conclusive evidence that long-term functional change and benefit are related to renal artery patency. This is why management of asymptomatic renal artery stenosis is primarily conservative/medical management. On the other hand there is no evidence that endovascular revascularization is superior to surgery in maintaining patency. Moreover the general impression from the findings of this study is that surgery is better for patients in terms of functional outcome and that surgical mortality, even from series carried out 15—30 years ago, was already not significantly higher than that for endovascular intervention. It is hoped that the findings from current large trials will help to formulate clear guidelines for

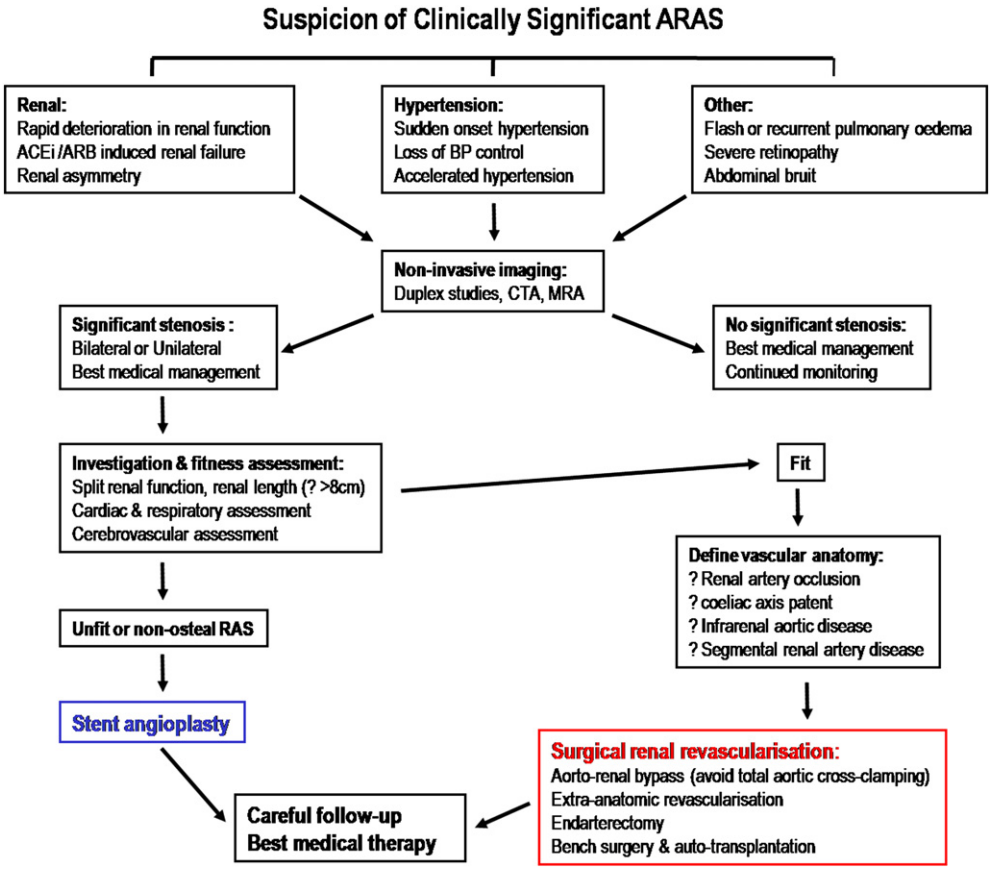

Figure 4 Suggested algorithm for management of patients with atherosclerotic renovascular disease.

indications for revascularization and promote patency to be re-addressed in future trials comparing the outcome of endovascular and surgical intervention in patients who would be *expected* to benefit from revascularization.

Conflict of Interest/Funding

None.

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